Amendment to the Claims:

Please amend the claims as follows:

Please cancel claims 6 and 7, 12, 28 and 40 to 43, without prejudice or disclaimer.

This listing of claims will replace all prior versions, and listing, of claims in the application:

Listing of Claims:

Claim 1 (currently amended): A transgenic mouse comprising:

a first transgenic nucleotide sequence, integrated into the genome of said mouse, comprising a sequence encoding the wild-type human amyloid precursor protein (hAPP) 751 amino acid isoform (hAPP751) operably linked to a first promoter; and

a second transgenic nucleotide sequence, integrated into the genome of said mouse, comprising a sequence encoding the **[[**a**]]** wild type human (h) α-synuclein operably linked to a second promoter;

wherein the first and second transgenic nucleotide sequences are expressed, the first and the second promoter comprises a neuron-active promoter, and as a result of expression of the hAPP and (h) α-synuclein, said transgenic mouse develops amyloidosis, neurofibrillary tangles and intraneuronal accumulation of (h) α-synuclein.

Claim 2 (original): The transgenic mouse of claim 1, wherein said first promoter comprises a platelet-derived growth factor β (PDGF-β) promoter.

Claim 3 (original): The transgenic mouse of claim 2, wherein a simian virus (SV)40 derived intron operably links said PDGF-β promoter to said first transgenic nucleotide sequence.

Claim 4 (original): The transgenic mouse of claim 1, wherein said first promoter comprises a Thy1 promoter.

Claim 5 (original): The transgenic mouse of claim 1, wherein said first promoter comprises a prion (PrP) promoter.

Claims 6 and 7 (canceled)

Claim 8 (original): The transgenic mouse of claim 1, wherein said second promoter comprises a Thy1 promoter.

Claim 9 (original): The transgenic mouse of claim 1, wherein said second promoter comprises a PrP promoter.

Claim 10 (original): The transgenic mouse of claim 1, wherein said second promoter comprises a PDGF-β promoter.

Claim 11 (original): The transgenic mouse of claim 10, wherein a SV40 derived intron operably links said PDGF-β promoter to said second transgenic nucleotide sequence.

Claim 12 (canceled)

Claim 13 (previously presented): The transgenic mouse of claim 1, wherein the nucleotide coding sequence of hAPP comprises an intron between exons 6 through 9 of the hAPP-encoding sequence.

Claims 14 to 26 (canceled)

Claim 27 (currently amended): A transgenic mouse comprising:

a first transgenic nucleotide sequence, integrated into the genome of said mouse, comprising a sequence encoding the wild-type human amyloid precursor protein (hAPP) 751 amino acid isoform (hAPP751) operably linked to a platelet derived growth factor β (PDGF-β) promoter operably linked to a simian virus (SV) 40 intron; and

a second transgenic nucleotide sequence, integrated into the genome of said mouse, comprising a sequence encoding **[[**a**]]** the wildtype human (h) α-synuclein operably linked to a PDGF-β promoter operably linked to an SV40 intron;

wherein the first and second transgenic nucleotide sequences are expressed, and as a result of expression of the hAPP751 and wildtype human (h) α-synuclein, said transgenic mouse develops amyloidosis, neurofibrillary tangles and intraneuronal accumulation of (h) α-synuclein.

Claims 28 to 31 (canceled)

Claim 32 (currently amended): The transgenic mouse of claim 27, wherein said transgenic mouse develops ~~neurodegenerative disease comprises~~ formation of intraneuronal inclusions characteristic of Lewy body disease.

Claim 33 (currently amended): The transgenic mouse of claim 27, wherein said transgenic mouse develops ~~neurodegenerative disease comprises~~ formation of fibrillary Lewy body-like inclusions.

Claim 34 (currently amended): The transgenic mouse of claim 27, wherein said transgenic mouse develops ~~neurodegenerative disease comprises~~ neuronal death.

Claim 35 (currently amended): The transgenic mouse of claim 27, wherein said transgenic mouse develops a ~~neurodegenerative disease comprises development of~~ motor deficit ~~deficits~~.

Claim 36 (currently amended): The transgenic mouse of claim 27, wherein age of onset of the amyloidosis, neurofibrillary tangles and intraneuronal accumulation of (h) α-synuclein ~~neurodegenerative disease~~ occurs at a significantly ($p<0.05$) younger age than in a singly transgenic (having only one of either the first or the second transgene) littermates.

Claim 37 (withdrawn - currently amended): A method for screening therapeutic agents for the prevention or treatment of neurological disease comprising

(a) administration of a therapeutic agent to the transgenic mouse of claim 1 ~~or claim 27~~; and,

(b) determining the effect of the therapeutic agent on the transgenic mouse.

Claim 38 (canceled)

Claim 39 (withdrawn - currently amended): A method for screening for an agent for the prevention or treatment of intraneuronal accumulation of α-synuclein, amyloidosis or neurofibrillary tangles, comprising

(a) providing a potential therapeutic agent;

(b) administering the potential therapeutic agent of (a) to the transgenic mouse of claim 1 ~~or claim 27~~; and

(c) determining whether because of the administering of the potential therapeutic agent in (b) intraneuronal accumulation of α-synuclein, amyloidosis or neurofibrillary tangles in the transgenic mice is prevented or slowed.

Claims 40 to 43 (canceled)

Claim 44 (new): A method for screening therapeutic agents for the prevention or treatment of neurological disease comprising

(a) administration of a therapeutic agent to the transgenic mouse of claim 27; and,

(b) determining the effect of the therapeutic agent on the transgenic mouse.

Claim 45 (new): A method for screening for an agent for the prevention or treatment of intraneuronal accumulation of α-synuclein, amyloidosis or neurofibrillary tangles, comprising

(a) providing a potential therapeutic agent;

(b) administering the potential therapeutic agent of (a) to the transgenic mouse of claim 1; and

(c) determining whether because of the administering of the potential therapeutic agent in (b) intraneuronal accumulation of α-synuclein, amyloidosis or neurofibrillary tangles in the transgenic mice is prevented or slowed.